

Chapter 16

Personality

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Introduction

Gordon Allport (1937) has penned some of the most influential lines in the history of personality research. He defined personality as the "... dynamic organization within the individual of those psychophysical systems that determine his unique [sic] adjustments to this environment" (p. 48) and that "... personality is something and personality does something" (p. 48). Together, these lines neatly summarize the primary mission of personality research: (1) the characterization of enduring qualities that give rise to regularities and consistencies in behaviour and the organization of these qualities and (2) how they achieve coherent functioning to actively adapt to the social environment (Livesley & Jang, 2005). As a result, much of mainstream personality research has been directed towards determining the number of basic traits, their organization, how they can be measured reliably, and the relationship between normal personality function and personality disorder.

The sheer volume of research on these questions has converged to show neuroticism (N), extraversion (E), openness to experience (O), agreeableness (A), and conscientiousness (C), popularly referred to as the "Big Five" (e.g. John & Srivastava, 1999), delineate the basic traits of normal personality. Although it would be incorrect to consider the Big Five as the definitive model of personality, its value cannot be underestimated because it serves as a useful framework in which to study personality. This is because the Big Five is related to competing personality models, such as Eysenck and Eysenck's (1975) psychoticism, extraversion, neuroticism model (PEN model) and Zuckerman, Kuhlman, Thornquist, and Kiers's (1991) alternative five-factor model in generally predictable ways. Moreover, it is of sufficient depth and breadth to subsume the ideas of alternate personality models (e.g. Aluja, Garcia, & Garcia, 2004; Larstone,

Jang, Livesley, Vernon, & Wolf, 2002; Markon, Krueger, & Watson, 2005).

Given reliability of the major personality measures and their well-understood relationship between one another, personality has been a target of behavioural geneticists for decades. Classic twin studies have yielded what is perhaps one of the most stable findings reported in the social sciences – additive genetic influences (h^2_A) account for between 40 and 50% of the total variability in personality, with nonshared environmental influences (e^2) accounting for the remainder and shared family effects (c^2) accounting for a negligible portion (e.g. Ando et al., 2002; Bouchard & Loehlin, 2001; Jang, Livesley, & Vernon, 1996; Livesley, Jang, Jackson, & Vernon, 1993). These findings have been consistently replicated across most of the popular inventories of normal and abnormal personality, awarding the psychological entity known as "personality" the status of a biologically based anatomical structure. As a result, personality inventories became the basis of several studies designed to identify putative loci for personality.

The Problem

However, something unexpected happened. Despite the stability of the phenotype and consistency of the heritability estimates, years of intense molecular genetic research has been unable to consistently identify the loci underlying any of the major personality traits (e.g. Munafò et al., 2003). One of the most famous examples is Ebstein et al.'s (1996) report that novelty seeking scores from the Temperament and Character Inventory (TCI) which was developed to operationalize Cloninger's "psychobiological model of temperament and character" (Cloninger, Svrakic, & Przybeck, 1993) was associated with the long form of the dopamine receptor D4 (DRD4) allele as predicted by the model. This association was followed by several independent replications but also non-replications by the same research group (e.g. Ebstein, Segman, & Benjamin, 1997; Ebstein,

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Gritsenko, & Nemanov, 1997). Several other research groups also attempted to replicate this association, and two meta-analyses of these reports concluded that the association was negligible (Kluger, Siegfried, & Ebstein, 2002; Schinka, Letsch, & Crawford, 2002). This inconsistency has been reported for other dopaminergic genes and personality measures. For example, no association between DRD4 or the D2 dopamine receptor (DRD2) gene and novelty seeking measured by the Multidimensional Personality Questionnaire (MPQ; Tellegen, 1982, unpublished manuscript) was found whereas a positive association was reported between DRD2 and a measure of novelty seeking (Berman, Ozkaragoz, McDonald, Young, & Noble, 2002).

Similar inconsistencies have been reported between genes regulating the serotonergic system and neuroticism. Several studies have reported an association between the short form of the 5HTT allele (that produces less serotonin transporter mRNA) and significantly increased NEO-PI-R neuroticism scores (e.g. Lesch et al., 1996) and related traits, such as harm avoidance (Osher, Hamer, & Benjamin, 2000) accompanied by a number of non-replications (e.g. Flory et al., 1998). Even when meta-analytic techniques are used, the results are mixed. Two meta-analyses concluded that the association was significant when neuroticism was assessed with the NEO-PI-R but not with TCI or related scales (Schinka, Busch, & Robichaux-Keene, 2004; Sen, Burmeister, & Ghosh, 2004), whereas another reached the opposite conclusion (Munafo, Clark, & Flint, 2005a; but see also Munafo, Clark, & Flint, 2005b). Presently, these inconsistencies have been attributed to methodological issues, including those of sample size and power. Current wisdom in the field is to overcome these limitations with brute force methods, such as designing studies that take advantage of different family relationships, testing increasingly more loci that are located closer together, and to recruit huge samples of subjects in the hope of aggregating effect size and averaging out error (see Plomin, DeFries, Craig, & McGuffin, 2003 for a discussion).

Studies with the purpose of identifying the unique experiences, milieu, or conditions that account for individual differences in personality have also largely come up empty-handed. This has been particularly surprising given that non-shared environmental factors consistently account for over half of the overall variability observed in most personality measures and their centrality in psychological and psychiatric theory. This failure is highlighted by Turkheimer and Waldron's (2000) extensive meta-analysis of the environmental research on personality. They found that differences in family constellation variables on average accounted for 1.1% of the variance; maternal and paternal behaviour accounted for 2.3 and 1.6%, respectively; sibling interaction accounted for 2.4% on average; and peer-teacher interactions accounted for 5.3% on average. In short, measured nonshared

environmental variables leave about 90% of the variance in personality and temperament unaccounted for. Furthermore, it should be noted that when they limited their analyses to studies that used a genetically informative design (e.g. twin and family studies), the effect size of measured nonshared environmental variables, on average, was halved!

The search for explanations for this state of affairs and their resolution will likely drive personality behavioural genetics for the next decade. A critical examination of some of the current issues in mainstream personality research and behavioural genetics suggests that perhaps the failure to identify putative loci and environment is because we may have "put the cart before the horse". That is, the molecular genetic research was undertaken on perhaps the naïve assumption that the personality phenotype was well understood. How might we have misunderstood personality? Gottesman and Gould (2003) pointed out that the more complex behaviour is the more genes are involved. Flipping the problem over, it also suggests that even if it is accepted that given the convergence of research that the Big Five represents the basic traits of personality, current measures of personality do not adequately capture its complexity.

For example, there remain disagreements as to which behaviours actually define each trait. Depue and Collins (1999) reviewed the definition of extraversion as assessed by the major scales. They found that all of the scales recognized sociability and affiliation but not all recognized agency (e.g. surgency, exhibitionism), activation (e.g. activity level), impulsivity – sensation seeking (e.g. novelty seeking, monotony avoidance), positive emotions (e.g. enthusiasm, cheerfulness), or optimism. Similarly, the NEO-PI-R neuroticism scale (Costa & McCrae, 1992) contains items assessing impulsive behaviours whereas these are not measured by the EPQ-R neuroticism (Eysenck & Eysenck, 1992), indicating that the definition of trait neuroticism is fundamentally different in each model. Thus, each measure can be reflecting the action of quite different genes.

Behavioural geneticists are now faced with two broad challenges to coming to understand this complexity. First, at the level of the phenotype, do current personality measures actually have sufficient bandwidth and fidelity to reflect personality? Second, do behavioural genetic methods or how they have been used sufficiently capture and reflect this complexity? In order to meet these challenges, we must first examine some of the recent findings in the behavioural genetics of personality and the issues they raise. Given the long association of behavioural genetics and personality there are many issues to consider that no short chapter such as this can adequately cover. As such, we have chosen to broadly discuss a few of the issues that we feel will be the source of some head-scratching over the next decade.

Current Topics

Using Genetics to Understand the Personality Phenotype

There is no doubt that personality is a complex phenotype that has required a huge effort to develop a systematic and rational approach to scale development to make personality accessible for research and clinical use. A good example of this systematic and rational approach is “the lexical models of personality”. This approach takes advantage of the natural language concepts used to describe behaviour. One source of such adjectives used in the past is the dictionary. Individuals are asked to rate themselves on each adjective (typically using a Likert scale of 1 “not like me at all” to 5 “very much like me”) and these ratings are subjected to factor analysis, with each extracted factor representing a personality “trait”. In fact, the “Big Five” basic personality traits mentioned earlier is the result of convergence of results from several lexical studies, more properly known as the five-factor model (FFM).

One of the most popular measures of the Big Five is the revised NEO personality inventory (NEO-PI-R; Costa & McCrae, 1992). Research using this inventory has shown that the five-factor structure and its psychometric properties are remarkably consistent across gender, age, race, and when translated into different languages, across cultures as well (e.g. Costa, McCrae, & Dye, 1991; McCrae & Allik, 2002; McCrae, Terracciano, & 78 Members of the Personality Profiles of Cultures Project, 2005). Given this stability, McCrae and Costa (1999) and McCrae (2004) proposed the five-factor theory (FFT) where they argued that the FFM structure is universal because the five personality traits are not affected by culture but solely shaped by biology that is common to human species.

Behavioural genetic methods, especially multivariate approaches, are well suited to test this hypothesis. Multivariate genetic analysis presumes that the observed correlation (or covariation) between two variables is mediated by both genetic and environmental factors shared by the two variables. The extent to which the two variables share genetic (i.e. pleiotropy) and environmental influences is indexed by the genetic (r_G) and environmental (r_E) correlation coefficients, respectively. Both r_G and r_E yield a coefficient that varies from -1.0 to $+1.0$ and is interpreted as any other correlation coefficient (e.g. Pearson's r) and can be subjected to factor analysis to determine the degree to which variables share a common genetic and environmental basis.

To test the basic premise of FFT, Yamagata et al. (2006) factored genetic and environmental correlations computed between all 30 of the NEO-PI-R facets using twin data from

Canada, Germany, and Japan. For each sample, the congruence coefficients between the genetic, environmental, and phenotypic factors ranged from 0.95 to 0.99, indicating that both genetic and environmental factors are responsible for the patterns of trait covariation observed in phenotypic analyses of trait structure. Comparison of genetic and environmental factors across three samples also revealed that the influence of genetic and environmental factors on trait covariance is similar across cultures.

These findings can be taken as supporting the validity of FFT, although it is unclear from this study what is genetically universal – the specific structure of the FFM or covariation of personality traits in general. We raise this issue because similar studies using different personality models also showed that by and large, no matter the phenotypic structure of traits (e.g. the model under study posits that two, three, four, or five factors) the number of genetic and environmental factors and patterns of loadings remain highly congruent (e.g. Carey & DiLalla, 1994; Livesley, Jang, & Vernon, 1998; Loehlin, 1982, 1987; Krueger, 2000; McCrae, Jang, Livesley, Riemann, & Angleitner, 2001). The fact that aetiological structure almost always resembles phenotypic structure no matter what that structure may be raises questions about how basic the Big Five are – revisiting the classic “number of factors question”.

An added complexity is that when one examines the personality literature most of the current research uses measures that have been developed using the concepts and language of one culture that was then exported to another. For example, the Yamagata et al. (2006) study used the NEO-PI-R which was developed using American usage of the English language and translated into German and Japanese. However, an imported scale may not capture all of the important personality characteristics found in another culture. There is a growing body of personality research using the lexical approach to analyse personality in other languages, such as Korean, Chinese, Japanese, Tagalog, and Croatian. A series of papers by Ashton and Lee (Ashton et al., 2004; Boies, Yoo, Ebacher, Lee, & Ashton, 2004; Hahn, Lee, & Ashton, 1999) has suggested a robust sixth factor describing honesty–humility or truthfulness. Also, Cheung et al. (2001) have shown that indigenous Chinese personality scales typically extract a sixth factor called “ren qing” (relationship orientation), reflecting harmony and face, personality features that do not generally appear in the English language, which were not well captured by the NEO-PI-R.

To add to the mix are the studies that fail to find the general congruence between phenotypic and aetiological structure. For example, Heath, Eaves, and Martin (1989) examined genetic and environmental structure of Eysenck's PEN model and found that the items comprising extraversion, neuroticism (and a fourth validity scale called Lie) scales were genetically and environmentally influenced via

single common latent factor per scale, whereas psychoticism factor showed unusual finding of zero heritability. In a subsequent study, Heath and Martin (1990) also reported that the phenotypic structure of psychoticism corresponded well to shared and nonshared environmental structure, but not to the genetic one, suggesting that psychoticism is an aetiologically heterogeneous construct. One obvious explanation is that the difference in findings simply reflects the natural characteristics of the population under study. A second more serious possibility is questions regarding the precision of the personality phenotype in general.

It is the latter question that is of most current interest to personality researchers. For example, let us return to look at Cloninger et al.'s (1993) "Psychobiological Model of Temperament and Character" and the measures developed to operationalize its tenets (the TCI and the earlier Tridimensional Personality Questionnaire; Cloninger, Przybeck, & Svrakic, 1991) in a little detail. This model has become very popular with behavioural geneticists because it has provided substantial guidance in the selection of candidate genes since the scales were developed to reflect the influence of specific biological processes. This model hypothesizes that personality is composed of four temperament traits and three character traits. The model posits that temperament traits manifest early in life, functioning as preconceptual biases in perceptual memory and habit formation. Each trait is hypothesized to be controlled by a unique genetically based neurotransmitter system: the dopaminergic system for novelty seeking (NS); the serotonergic system for harm avoidance (HA); and the noradrenergic system for reward dependence (RD). A fourth dimension labelled "persistence" has been suggested (Cloninger et al., 1993) but no putative neurotransmitter system has been hypothesized.

The three character dimensions are self-directedness (SD), cooperativeness (CO), and self-transcendence (ST), which are hypothesized to be traits that reflect learned, maturational variations in goals, values, and self-concepts that develop in adulthood through conceptual or insight-based learning. As such, character traits should show little heritable influence in contrast to temperament traits. Despite the model's theoretical strength, psychometric examination of the TCI scales themselves has raised several questions regarding their reliability (Gana & Trouillet, 2003; Stewart, Ebmeier, & Deary, 2004). Straightforward behavioural genetic analyses have also questioned the theory and scales. First, contrary to predictions, the character traits have been found to be substantially heritable¹ (Ando et al., 2002; Gillespie, Cloninger, Heath, & Martin, 2003), and molecular

genetic studies have found that 5-HTTLPR gene was associated with cooperativeness, but not with temperament traits (e.g. Kumakiri et al., 1999).

Ando et al. (2004) examined genetic and environmental factor structure of TCI and used the findings of multivariate genetic analyses to reorganize the content of the TCI scales. Each of the TCI dimensions, like most personality scales, is composed of several narrow sub- or "facet" traits. For example, NS is defined by exploratory excitability, impulsiveness, extravagance, and disorderliness. The genetic correlation between all of the facet traits defining NS, HA, and RD dimensions on a sample of 414 pairs of MZ and 203 DZ twin pairs from Japan was factored (see Table 16.1). Factor analysis of the genetic intercorrelations yielded factors that did not quite resemble the phenotypic structure of NS, HA, and RD. As shown in Table 16.1, only the subtraits defining reward dependence (factor II) lined up as originally designed. Using this information, harm avoidance (r-HA) was revised to consist of (low) exploratory excitability, anticipatory worry, fear of uncertainty, shyness, and fatigability. Novelty seeking (r-NS) was revised to consist of impulsiveness, extravagance, and disorderliness, and RD was unchanged. The genetic and environmental correlations between r-NS, r-HA, and RD were very small (ranging from -0.02 to 0.11), indicating that the revised temperament scales were rendered genetically homogeneous and independent. What is important about these results is that they suggest that the genotyping research based on TCI dimensions as originally designed is reflecting several, possibly competing, influences. Thus, if the primary phenotype was the total dimension score, it would be unclear as to what specific personality trait is actually associated with the gene.

This issue highlights one research agenda for behavioural genetics – the revision of scales to be more genetically homogeneous. Genetic correlations are not only useful in revising

Table 16.1 Varimax rotated principal factor analysis loading matrix of the genetic correlations estimated between the TCI temperament subscales

	I	II	III	IV
Novelty seeking				
Exploratory excitability	0.62	0.28	0.25	0.29
Impulsiveness	-0.10	0.03	0.03	0.76
Extravagance	0.02	0.16	0.00	0.72
Disorderliness	0.03	-0.16	0.15	0.74
Harm avoidance				
Anticipatory worry	-0.87	0.01	-0.03	-0.01
Fear of uncertainty	-0.51	0.28	-0.32	-0.43
Shyness	-0.76	-0.16	-0.22	-0.05
Fatigability	-0.72	-0.26	-0.06	-0.10
Reward dependence				
Sentimentality	-0.06	0.56	0.60	-0.04
Attachment	0.04	0.70	0.00	0.22
Dependence	-0.09	0.86	-0.19	0.13

Source: Ando et al. (2004).

¹ Cloninger conceded that character traits are as heritable as temperamental traits, yet have different biological base than temperament traits (personal communication, July, 2005; see also Gillespie et al., 2003).

content, but they can also be used to address basic psychometric issues such as reliability. For example, this approach can be easily applied to the correlations between items to index the internal consistency of personality scales as well as test-retest reliability. Genetic (and environmental) correlations between scores of a personality scale measured at different time intervals (e.g. longitudinal twin study) provide information regarding the extent to which a trait is consistently influenced by the same genes across time (e.g. Gillespie, Evans, Wright, & Martin, 2004). Genetically reliable personality measures are suitable for linkage/association study because it provides more statistical power to detect genes. It should be also noted that similar logic could also be applied to the use of environmental correlations, which can help study searching for the specific environmental factors influencing personality.

Another recommendation for future research is to use genetic/environmental factor scores for linkage/association studies. Sham et al. (2001) recently described a basic process that enables a score on any measure, like neuroticism, to be split into two scores: to reflect pattern of genetic influences and the other environmental influences. The basic approach is to derive a weight for each of the genetic and environmental effects that can be applied to the phenotypic score of an existing inventory. These weights function much like weights used to compute factor scores but instead of being derived from the phenotypic correlations between variables, they are derived from matrices of genetic and environmental correlations.

Theoretically, it is possible even to break the genetic score further into separate scores that reflect the variability in neuroticism due to different sets of genes. These genetically and environmentally indexed scales would reduce genetic/environmental noise in a measure of a behaviour when searching for specific environment/genes influencing the trait and increase the power to find them (Lander & Botstein, 1989), as was reported in several simulation studies and studies using actual data (e.g. Eaves & Meyer, 1994; Cardon, Smith, Fulker, Kimberling, Pennington, & DeFries, 1994; Boomsma, 1996; Boomsma & Dolan, 1998). For clinicians, such scales would be also extremely useful as they could reflect the level at which psychotherapeutic or pharmacological treatments are acting.

What Is Inherited?

Genetic and environmental factor analyses discussed above are simple forms of what is called the “independent pathway (IP) model” illustrated in Fig. 16.1. In this model, higher-order constructs (e.g. neuroticism) simply reflect sum total of the pleiotropic action of genes and environmental influ-

ences shared by all lower-order traits rather than the effects of a phenotypic entity. Thus, under an IP model, higher-order constructs are, in effect, relegated to the status of a convenient heuristic device to label the covariance of traits.

A more stringent multivariate genetic model is the “common pathway (CP) model” illustrated in Fig. 16.2. The centre section of this figure is identical to the form of the contemporary factor analysis model. The addition of genetic and environmental influences to the latent variable P (note that the existence of P is unmeasured and is inferred by the degree to which the measured variables appear together), which represents a higher-order trait, transforms the latent variable into a veridical entity that has a basis in biology. The most important aspect of this figure is that P mediates 100% of the covariance of the lower-order traits that define it and that each of the lower-order traits is best understood as exemplars of the higher-order construct. Although both IP and CP models hypothesize that personality trait facets share a common genetic basis, the primary difference is that in the IP model, *no* independently inherited P is required to explain the covariation between personality measures. Knowing whether personality is structured like an IP or CP model is fundamentally important because each suggests quite different approaches to the search for putative genes. For example, if the CP model is correct, this implies that there are specific genes for the higher-order construct or domain. As such, as in current approaches, the phenotypic factor score could be used because each facet is an exemplar of the domain that has a common genetic basis with all other aspects of the domain. In contrast, if the IP model is correct, it would mean that either the total summative score across facet traits or phenotypic factor score would not be ideal because it could reflect different, possibly competing, aetiological influences. Rather, a phenotypic factor score based on only those subsets of facets that share a common genetic basis can be associated with various candidate genes (or genetic factor score as described above can be used).

It is important to note that the IP and CP models illustrated in Figs. 16.1 and 16.2 appear equivalent because every facet trait is shown to share a common genetic and environmental basis with the others defining each domain. However, in the CP model it must be remembered that because of the latent phenotype P, *all* facets must share a common genetic and environmental basis. When this is not the case, for example, when one of the facets may not share a common genetic basis with the other facets, the CP model is effectively reduced to the IP model in which that path between the common genetic factor G and the affected facet trait is set to zero. The observed covariance of the affected facet with the others is maintained by the environmental influences in common. Thus, it should be clear that the CP and IP models provide quite different explanations for why facet traits covary.

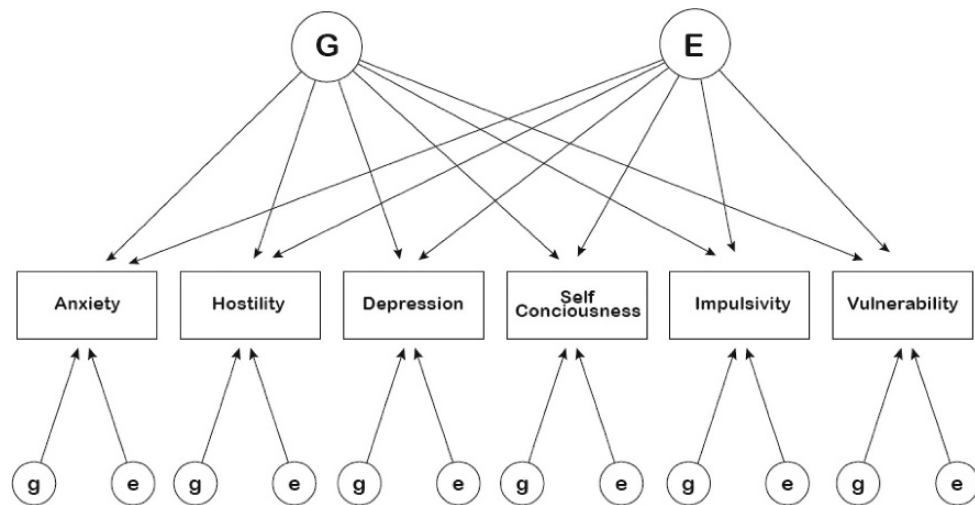


Fig. 16.1 Independent pathways model

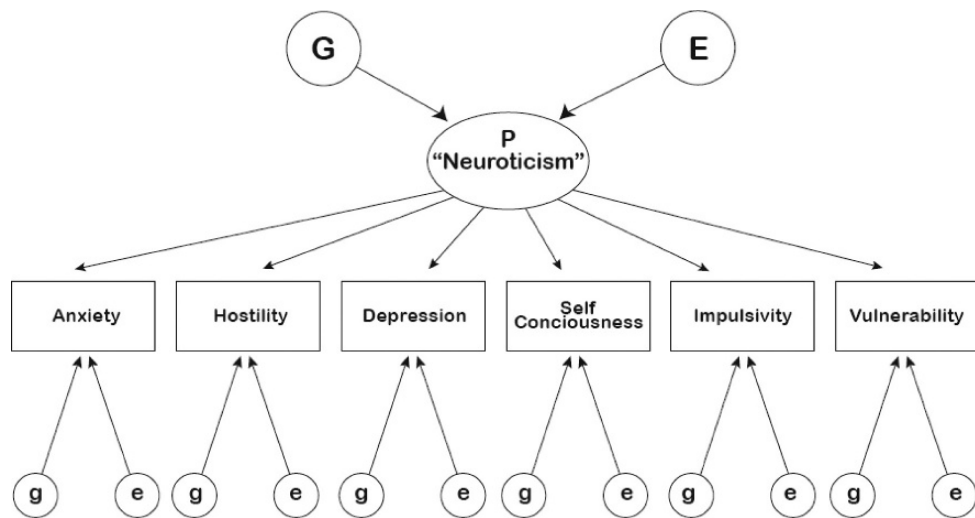


Fig. 16.2 Common pathways model

The relative correctness of the IP or CP models has important implications for personality theory. It speaks of the validity of higher-order traits such as N, E, O, A, and C as veridical entities.

Despite the importance of these structural models for personality research and theory, current research using IP and CP models has yet to provide a clear indication as to how personality is organized. For example, Jang et al. (2002) fit both CP and IP models to the six facets defining each of the NEO-PI-R domains and the CP model was rejected for all domains. Instead, an IP model specifying two additive genetic factors and two nonshared environmental factors provided the best fit. This finding suggests that there are *no* genes for N, E, O, A, and C per se. Rather, as noted earlier, higher-order traits are simply a convenient heuristic to describe the action of genes. This conclusion is consistent with the lex-

ical view of Saucier and Goldberg (1996), who argued that the five domains are merely a convenient way of organizing lower-order traits. In contrast, Johnson and Krueger (2005a) reported that for the adjectives describing neuroticism and extraversion, a CP model provided a good fit, and similar to Jang et al. (2001), for the adjectives describing agreeableness, conscientiousness, and openness, the IP model provided the best fit. This finding supports the traditional view that N and E are inherited entities and can be treated as such.

However, it should be noted that in these two papers, the decision to retain the IP or CP model was based on what could be called “flexible” statistical grounds. For example, by accepting a slightly different cut-off point for a fit index, such as 0.08 as opposed to 0.05, either the CP or IP model provides a satisfactory fit. Similarly, both papers used

different fit indices to make their decisions. It is quite possible that electing to place emphasis on one fit index over the other (e.g. Bayesian information criterion versus the root mean square residual) can yield quite different conclusions.

In fact, in current research it is quite common to find that *both* the CP and IP models provide a satisfactory explanation for the data. To illustrate the phenomenon, consider Young, Stallings, Corley, Krauter, and Hewitt's (2000) genetic analysis of adolescent behavioural disinhibition. In this study of DSM-IV symptom counts for conduct disorder, attention deficit disorder, substance experimentation, and novelty seeking, a one-factor CP model and a one genetic factor IP provided a good fit to the data. The reported $\chi^2_{\text{difference}}$ between the models was a non-significant ($p > 0.05$) 1.51. As a result, Young et al. selected the common pathways model because "...this is a more parsimonious model than the independent pathway model and shows no significant decrement in fit by χ^2 difference test..." (pp. 690–691). Because statistical guidance was lacking, the authors picked the simplest model. However, is the CP model actually the simplest? It adds complexity in that it trades off simplicity for stringency because it specifies that the trait covariation is entirely due to the mediating action of a higher-order variable. It can be argued that being able to drop the requirement of having to invoke an unmeasured higher-order construct renders a simpler explanation for the data.

At the present time, it appears that when statistical guidance is lacking, the choice of model can become rather arbitrary. In the meantime, the easiest way to help resolve the problem is to conduct replication studies, use of larger sample sizes, and power calculation to help gauge the veracity of the results.

Role of Personality Theory

Theories of personality and personality development in mainstream personality research have tended to take a backseat to efforts focused on developing reliable measures that had tended to use an empirical approach (e.g. lexical approach). However, this has caused a disconnect between trait models of personality and concepts used to describe personality and its development (Digman, 1997). For example, do any of the major personality domains assessed by any of the major inventories capture the concepts from Freud's theories on psychosocial development?

Why is this important? Some may argue that these theories are only of historical interest to personality psychologists who would not expect behavioural genetic or molecular genetic studies to address the constructs of classical personality theory. We disagree. Personality theory is the source of testable hypotheses regarding personality development,

the relationship between different traits, its structure, and the role personality plays in everyday living and mental illness. Human beings strive for "personal growth" or "self-actualization" – not just to be more or less extraverted. Such striving implies the interplay of personality traits from several domains, such as extraversion and openness to experience. The real question for personality research is how do trait concepts come together (genetic? environmental? interplay?) through which "self-actualizing" tendencies develop and are expressed? Behavioural genetics can help address these basic questions just as they did for trait models. Moreover, in light of our previous discussion on model selection, theory is a means to guide the selection of model when statistical criteria are ambiguous.

There is a growing body of research along these lines. For example, phenotypically, Digman (1997) has shown that two higher-order factors have been consistently extracted from the Big Five personality factors (Digman, 1997). The first he called alpha (α) which was typically defined by factor loadings from agreeableness, conscientiousness, and neuroticism which encompass aggression, hostility, impulse restraint, and neurotic defence akin to Freud's theories on psychosocial development or Adler's "social interest". Similarly, the emergence of extraversion and openness to experience as a single factor (β) is a far more comprehensive reflection of constructs such as Rodger's "personal growth", Adler's "superiority striving", or Maslow's "self-actualization" than is possible by either trait alone. He argued that such higher-order traits are important because they provide a tangible link between psychometric models used to develop reliable taxonomies and measures to theories of personality development.

Despite the apparent increase in conceptual clarity afforded by α and β , their actual relationship to developmental concepts has not been evaluated empirically. Such work cannot be conducted until the stability of these higher-order traits is better established. A recent paper by Jang et al. (2006) has begun to address this issue by conducting multivariate genetic analyses of the five NEO-PI-R domain inducting to determine if the α and β constructs can be reliably reproduced across a diverse range of independent samples. They report that two CP models could explain the covariance of N, E, O, A, and C from twin samples drawn from Canada, Germany, and Japan. The first model broadly represented α and the second resembled β .

Of particular importance is the finding that although the domains share some genetic and environmental influences to differing degrees, a great deal of the variability of domains is due to genetic and environmental factors *unique* to each facet. For example, Jang, McCrae, Angleitner, Riemann, and Livesley (1998) estimated the heritability of the 30 NEO-PI-R facet traits after all genetic influence due to the

higher-order traits was removed using regression techniques. Substantial residual heritability was found for each trait that accounted for between 25% (competence) and 65% (dutifulness) of the variance. Livesley et al. (1998) also reported similar findings for personality disorder traits. These findings illustrate the oft forgotten fact that not all domains play equally important roles in personality when in practice, the opposite is often assumed. Personality research often tends to focus on what is common between traits when in fact, genetic and environmental factors specific to each facet or domain account for a greater proportion of the total variability. As such, when working with personality scale scores, it is not clear at which level the genes and environmental influences are exerting their influences; certain genes may contribute to facet-specific genetic variance, perhaps even item-specific variance, whereas other genes contribute to the variability of the domains or higher-order factors such as α . A simple recommendation for association study is to examine effects of specific genes/environment at all levels.

Consistency of Heritability Estimates

The chapter opened with a pronouncement that additive genetic and nonshared environmental influences accounted for all of the variability in personality measures. However, there exist several studies which suggest that not all of the genetic variability is additive (e.g. Loehlin, 1986, 1992; Loehlin, Horn, & Willermann, 1997; Loehlin & Nicholls, 1976) but non-additive (dominance or additive-to-additive epistasis effects; symbolized as d^2). There are a number of potential explanations for these findings, some of which are methodological and some of which causes one to pause and think about the inclusiveness of the major models of personality. For example, one possibility is that the presence of these effects is a reflection of content unique to specific personality scales such as the MPQ (Waller & Shaver, 1994), the TCI (Cloninger et al., 1993), or the Revised Eysenck Personality Questionnaire (EPQ-R; Eysenck & Eysenck, 1975), as reported in Keller, Coventry, Heath, and Martin (2005), in contrast to studies using the NEO-PI-R (e.g. Jang et al., 1996, 1998; Riemann, Angleitner, & Strelau, 1997) in which d^2 effects are rarely, if ever, found. This suggests that any one personality inventory alone does not necessarily provide a comprehensive assessment of personality.

The presence of non-additive effects may not be a personality measurement issue per se, but rather attributable to the possibility that behavioural genetic methods are not sensitive enough to reliably detect these effects. This is suggested by the reports of non-additive effects inconsistently found on the same measures (e.g. Ando et al., 2002; Eaves, Eysenck, & Martin, 1989; Gillespie et al., 2003;

Heiman, Stallings, Hofer, & Hewitt, 2003). This is because additive and non-additive genetic effects (both epistasis and dominance) are confounded in a twin-reared-together design, and only when large sample size is used and other genetically informative relationships (e.g. parents or non-twin siblings) are incorporated into the twin-reared-together design are non-additive effects reported more consistently (Eaves et al., 1999; Eaves & Carbonneau, 1998; Finkel & McGue, 1997; Keller et al., 2005; Lake, Eaves, & Maes, 2000). Several association studies showing that only combination of several genes can explain variance in personality suggest that non-additive effects are an important influence in personality (e.g. Benjamin, Osher, Kotler, et al., 2000; Benjamin Benjamin, Osher, Lichtenberg, et al., 2000; Strobel, Lesch, Jatzke, Paetzold, & Brocke, 2003).

Methodological issues also impact the magnitude of genetic effects. Although heritability in the 30–50% is typically observed for self-report measures, when multiple peer ratings or both self- and peer ratings are subjected to behavioural genetic analysis, h^2_A has been found to account for between 60 and 80% of the total variability (e.g. Heath, Neale, Kessler, Eaves, & Kendler, 1992; Riemann et al., 1997; Wolf, Angleitner, Spinath, Riemann, & Strelau, 2004). Multiple peer ratings can control rater-specific bias, which are included in estimates of e^2 , and thus are considered more objective than self-report. Thus, inflated rates of measurement error may be an important contributing factor for the disappointing results of studies designed to find sources of e^2 because it is possible that a significant proportion (50–70%) of nonshared environmental variance typically observed for classical twin studies may largely reflect measurement error and not actual environmental variation.

Another issue regarding the consistency of the heritability estimates is gene–environment interaction. If one thinks about what the heritability coefficient represents, one could say it represents (1) a snapshot of the magnitude of genetic effects on any sample and (2) the average genetic effect over all conditions. Thus, it could be that all research is correct, and the differences in results – be they variations in values of h^2 or type of genetic effect, h^2_A , h^2_D , c^2 – simply reflect the fact that different genes are in action at different times or in the face of certain environments. Personality theory has always stressed that events in childhood are important in personality development and future psychopathology. Despite the ubiquity of primacy of early experience in many of the theories of psychopathology, the primacy of early experience has a weak evidence base (e.g. Paris, 2001). For example, in the personality disorders a large body of research (e.g. Garnezy & Masten, 1994) shows that negative childhood experiences need not necessarily lead to psychopathological outcomes in adult life. As a result, theorists suggest that adversities in combination with genetic liabilities during

development increase the risk for mental disorders. Within the medicine and the social sciences this process of gene–environment interplay is called the diathesis–stress model of illness. This model in some form is usually invoked to explain why despite the fact that many people may carry the genes for mental illness, or may have experienced some kind of horrible trauma, not all of them will develop mental illness. In its simplest form, the model postulates that adverse experiences interact with an underlying genetic liability that leads to disease.

However, this explanatory model is very broad and does not specify the mechanisms of gene–environment interplay. Behavioural genetics has provided the means to test and delineate the mechanism of action. One such mechanism is genetic control of exposure to the environment in which genetic factors influence the probability of exposure to adverse events (Kendler & Eaves, 1986). In some fields, this phenomenon is referred to as an “amplification effect” (e.g. Paris, 1994, 1996) but within behavioural genetics it is called genotype by environment correlation – the extent to which individuals are exposed to environments as a function of their genetic propensities. Three general types of genotype–environment correlation have been hypothesized (see Plomin, DeFries, & Loehlin, 1977; Scarr & McCartney, 1983). The first is passive genotype–environment correlation that occurs because children share heredity and environments with members of their family and can thus passively inherit environments correlated with their genetic propensities. The second is called reactive in which the experiences of the child are derived from reactions of other people to the child’s genetic propensities. The third is the active type that occurs when children actively select or create environments commensurate with their underlying genetic propensities.

The other interplay effect is environmental moderation of genetic and environmental variability referred to as gene–environment interaction (Plomin, DeFries, and McClearn, 1990). The importance of gene–environment interaction has been shown by several molecular genetic studies. For example, Caspi, McClay, and Moffitt’s (2002) study of the development of antisocial behaviour is particularly salient to this discussion. Clinical research has identified childhood abuse, such as erratic, coercive, and punitive parenting, as one of the major risk factors for the development of antisocial behaviour in boys, and the risk for conduct disorder increases the earlier the abuse begins. However, there is often little 1:1 correspondence between environmental conditions and phenotype, so the presence of a genetic liability for the disorder must be involved. In the case of antisocial behaviour, the monoamine oxidase A gene (MAOA gene Xp11.23-11.4) was selected because it has been associated with aggressive behaviour in mice as well as some human studies.

This sample consisted of 1,037 children who had been assessed at 9 different ages for levels of maltreatment (no, probable, and severe maltreatment) and MAOA activity (low or high). They found that the effect of maltreatment was significantly weaker among males with high MAOA activity than those with low activity. Moreover, the probable and high maltreatment group did not differ in MAOA activity, indicating that the genotype did not influence exposure to maltreatment. These results demonstrate that the MAOA gene modifies the influence of maltreatment. Similar dramatic gene–environment interaction effects have been shown for genes believed implicated in depression (e.g. Caspi et al., 2003; but see also Eaves, 2006, for possibility of statistical artefacts in these studies).

For classical twin studies, there is a growing body of literature demonstrating that heritability varies over environmental condition. For example, Jang et al. (2005) showed that perceived levels of family conflict and maternal indulgence moderated the genetic influences underlying emotional instability, a trait delineating personality disorder, and is related to neuroticism. Specifically, this study found that the estimates of h^2_A varied between 70 and 44% over levels of maternal indulgence and 92 and 15% over levels of family conflict. Moderation was not limited to genetic effects, shared family environment effects (c^2), and ranged from 23 to 40% and 30 to 44% over levels of maternal overprotection and paternal care, respectively. This final set of results suggests that the apparent lack of c^2 is the result of its effects being averaged out over different conditions and that they might be cumulative. Moreover, it also highlights the fact that environment–environment interaction or experience by environment interaction is just as important. Such models allow us to test hypotheses about how some people can live in the most adverse conditions (e.g. extreme poverty) but display no ill effects. Is it because of the presence of another environmental factor, such as a caring mother who attends to the emotional needs of a child, cancelling out the influence of poverty?

There are an increasing number of studies reporting gene–environment interactions. Recently, Button, Scourfield, Martin, Purcell, and McGuffin (2005) observed that increasing genetic influences on antisocial behaviours with a concomitant decrease in c^2 as level of family dysfunction increased in samples of 5- to 18-year-old children. Boomsma, de Geus, van Baal, and Koopmans (1999) observed that genetic influences on disinhibition were smaller and shared environmental influences were larger for male adolescents raised in a religious family than for those raised in a non-religious family. Turkheimer, Haley, Waldron, D’Onofrio, and Gottesman (2003) observed that among 7-year-old children, genetic influences on intelligence were larger and shared environmental influences were smaller for those in more impoverished families (see also

Dick, Rose, Viken, Kaprio, & Koskenvuo, 2001; Johnson & Krueger, 2005a, 2005b). It is clear that the study of interplay effects is vitally important for personality research. Studies of this kind may be the key to understanding how personality develops and explain the inconsistencies in the current behavioural genetic literature.

What Does Personality Do?

Allport (1937) also noted that “personality does something”. Basically, personality is a general disposition that governs the way individuals normally behave across situations, and current research in this area has tended to focus on the role of personality on mental illness. Clinical studies consistently find that personality features are correlated (i.e. comorbid) with virtually all forms of common psychopathology. For example, individuals with depression frequently report being anxious (Kendler, 1996; Zimmerman, Chelminski, & McDermt, 2002). Further, personality features have been delineated as diagnostic criteria in many forms of psychopathology. For example, the diagnostic criteria for DSM-IV major depressive episode (APA; 1994) includes “feelings of guilt and worthlessness”, item content typically found in measures of trait neuroticism.

A review of the personality literature suggests that personality impacts psychopathology in three ways (Jang et al., 2006). The first hypothesizes that personality factors increase the risk for developing psychiatric disorder. In this model, both personality and psychopathology are qualitatively distinct entities; however, certain personality dimensions alone or in combination with others increase the likelihood of developing psychiatric disorder (e.g. Metalsky, Halberstadt, & Abramson, 1987). The second hypothesizes that personality and psychopathology occupy a single domain or spectrum and psychopathology is simply a display of the extremes of normal personality function (Eysenck, 1994). For example, Trull, Waudby, and Sher (2004) reported that DSM-IV cluster B personality disorder symptoms (particularly antisocial and borderline symptoms) were consistently associated with alcohol use disorders in a non-clinical sample of 395 young adults but also significantly associated with alcohol use disorders above and beyond what was accounted for by normal personality traits. This suggests that personality disorder symptoms predict unique variance in substance use disorders that clearly reflects maladaptive aspects of personality. The third hypothesis is that personality plays a minor role in the development of disorder and changes in observed personality are simply the result of disorder. For example, although minor personality changes are frequently observed before the onset of major depressive disorder, major personality

changes occur after onset (e.g. Chien & Dunner, 1996; see also Goldsmith, Lemery, & Essex, 2004; Widiger, Verheul, & van den Brink, 1999, for a similar classification).

Little, if any, current behavioural genetics research has directly investigated these possibilities. Rather, research has largely been descriptive, tending focus on showing that the comorbidity of personality and psychopathology is partially due to a common genetic basis. However, current findings provide some circumstantial evidence for the spectrum model of disorder. For example, Krueger (1999) reported that the phenotypic comorbidity of 10 DSM-III-R common mental disorders can be arranged into 2 higher-order constructs that describe disorders directed inward towards oneself (“internalizing” such as depression) as opposed to disorders that are directed outward (“externalizing” such as antisocial personality). In a subsequent study, Krueger et al. (2002) fitted a variety of genetic models to the data and found that a one-factor common pathway genetic model (similar in form to Figs. 16.1 and 16.2) provided the best explanation for the covariation of these measures, suggesting that the externalizing disorders are inherited as a single genetically based syndrome. However, despite Krueger et al.’s (2002) report that the fit of a one-factor common pathways model provided a superior fit to any model (e.g. models that did not include P), as discussed earlier, some lingering doubts remain as to whether this model truly provides the best explanation. For example, Kendler, Prescott, Myers, and Neale (2003) found that the internalizing and externalizing factors are not inherited as a single genetically based syndrome at all. They showed that an independent pathways model (of the form illustrated in Fig. 16.1) in which multiple genetic and environmental factors directly influenced each disorder provided a far superior fit to their data.

So which is correct? Are syndromes inherited as a unitary entity or are they convenient names for frequently comorbid disorders that share a common genetic basis? Once again, these kinds of results highlight important strengths and weaknesses of the behavioural genetic models and approaches used to study comorbidity. On the one hand, the models explain why comorbidity exists and provides, in principle, the means to study the organization of variables. As discussed earlier, what the current research has highlighted is our reliance on statistics and fit indices to make our decisions. This is not a new idea, because as shown quite often in the literature, such indices alone are insufficient to make a decision.

Directions for the Future

The current behavioural genetics of personality research has been extremely important in bringing to light a number of issues concerning what personality is and what personality

does. We have also highlighted broad directions where more research needs to be spent. The remainder of this chapter will present some potential ideas to resolve these issues. A theme of this chapter is the complexity of the personality phenotype. Another approach that might provide some insight into disentangling this complexity is to develop personality endophenotypes (neurobiological correlates of personality) and use these to validate existing scales (see Gottesman & Gould, 2003). This approach has been useful in alcohol research. For example, Hesselbrock, Begleiter, Porjesz, O'Connor, and Bauer (2001) noted that a difficulty with genotyping studies is that the clinical heterogeneity of the disorder results in a poorly defined phenotype for genetic analysis and that better results may be obtained by switching to diagnostic endophenotypes.

One endophenotype that has received considerable attention is the P300 event-related brain potential (ERP) waveform. ERPs are recordings of neuro-electrical activity in response to stimuli recorded by electrodes on the scalp. Common stimuli include flashing lights, reactions to a short emotionally laden video clip, or noises (beeps, etc.). This endophenotype has been shown to be a valid neurobiological correlate of alcoholism in both males and females (Prabhu, Porjesz, Chorlian, Wang, Stimus, & Begleitner, 2001). Hesselbrock et al. (2001) found significant reductions in P300 amplitude between alcoholics and non-alcoholics, between unaffected relatives of alcoholics and relatives of controls, and between unaffected offspring of alcoholic fathers and offspring of controls. Almasy et al. (2001) conducted a genome-wide scan of P300 responses to a semantic priming task on 604 individuals in 100 pedigrees. They showed that the P300 waveform was significantly heritable (40–50% range) and reported significant evidence of linkage to chromosome 5 and suggestive evidence of linkage to chromosome 4.

Within personality research, a promising endophenotype suitable for linkage/association study is frontal electroencephalographic (EEG) asymmetry (Allen & Kline, 2004; Davidson, Jackson, & Larson, 2000). Frontal EEG asymmetry represents balance between cortical activity in left and right frontal regions; more specifically, a frontal EEG asymmetry score is obtained by subtracting log-transformed power density value in the alpha band (8–13 Hz) in the left frontal cite (e.g. F3) from the right frontal cite (e.g. F4). Because alpha power is inversely associated with cortical activity, the positive score indicates greater left-sided activity (see Tomarken, Davidson, Wheeler, & Doss, 1992). Previous research has shown that greater left-sided activity was consistently associated with stronger behavioural activation system (i.e. sensitivity to reward; Gray, 1987) and weaker behavioural inhibition system (i.e. sensitivity to punishment; Gray, 1987; Coan & Allen, 2003; Harmon-Jones & Allen, 1998; Sutton & Davidson, 1997), more positive and

less negative mood (Tomarken et al., 1992), higher reactivity to positive stimuli and lower reactivity to negative stimuli (Tomarken, Davidson, & Henriques, 1992; Wheeler, Davidson, & Tomarken, 1993), and reactivity to maternal separation among infants (Davidson & Fox, 1989). Lastly, a pilot study observed that frontal EEG asymmetry was heritable and genetically associated with negative emotionality among 66 female twin pairs (Coan, Allen, Malone, & Iacono, 2003).

For those interested in what personality may do, behavioural genetic methods provide the means to determine the role(s) any specific personality variable plays in a disorder. As discussed earlier, does variation in trait anxiety increase the vulnerability to depression, or is depression the extremes of normal anxiousness, or are changes in anxiety simply a consequence of depression? Current behavioural genetic methods provide some avenues to explore these alternatives. A straightforward way to investigate if personality acts as a risk factor is to apply models of gene–environment correlation and gene–environment interaction. The validity of a personality-as-risk-factor model rests on demonstrating comorbidity that is not contemporaneous but veridical by being caused by a shared aetiology estimated by r_G and r_E . Second, it depends on some form of gene–environment correlation – that is, genetically based personality traits help create or modify environments suspected to increase risk for a particular psychopathology. The third step is to demonstrate that the environment that personality helped shape “triggers” for the onset of another genetically based disorder. For example, people with high levels of genetically based sensation seeking prefer an urban as opposed to rural lifestyle where alcohol and other substances are readily available. The accessibility of alcohol could trigger the onset of genetically based alcoholism via the mechanism of gene–environment interaction.

A test for a personality-psychopathology spectrum is that personality and disorder share a common aetiological basis. However, one possible way to differentiate the risk and spectrum models is the degree to which personality and the other disorder share a common genetic basis and how this shared aetiology is structured. What those conditions are would be a fascinating research process. Finally, it is important that time is included in these tests. It is clearly the most crucial element to determine if personality is the cause or effect of psychopathology. To test if personality is simply a consequence of other mental illnesses, it must be shown that significant personality changes do not predate the onset of disorder. However, this may not be enough. Recall that quantitative genetic theory states that the phenotype represents the sum of genetic and environmental action, and it should be clear that genetic factors underlying personality can substantially increase the risk for the development of another disorder, although not reflected in the phenotype. As such, a significant genetic correlation can exist between two variables,

but because environmental factors can work in the opposite direction (that is, r_E is negative) the sum of these effects as reflected in the phenotypic correlation is zero (Carey, 2003), suggesting that the two variables are unrelated when in fact they are related.

Thus, it becomes extremely important to study genetic and environmental influences in the context of time. Current longitudinal behavioural genetic research has shown that different aetiological factors can operate at different times during development as shown by the well-known Nonshared Environment in Adolescent Development project (Neiderhiser, Reiss, & Hetherington, 1996) which has shown that genetic influences were important for both change and stability in antisocial behaviour.

In summary, the ability of behavioural genetic methods to move beyond the phenotype to focus on aetiology has been extremely important in supporting long-held assumptions or has shaken the foundations of issues long thought resolved in traditional personality research. It has also certainly minimized the centrality of some long-debated issues, such as the number of domains debate, but has renewed issues over content and definition of the domains. It has also begun to force an integration of personality measurement and personality theory. The goal of behavioural geneticists working in personality is to continue to apply these methods to resolve these issues. That is, these methods should be applied to issues important in personality research such as to aid in scale development and revision as opposed to uncritically accepting current conceptions of personality. It is hoped that the ideas in this chapter will stimulate thought and provoke a more thorough integration of personality and behavioural genetics.

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